Acute Kidney Injury In Polytrauma Patient

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- **A 36-year-old man is evaluated in the ER after being trapped for 20 hours under a collapsed concrete wall. On physical examination, pulse rate is 100/min and B.P 112/50 mm Hg. On pulmonary examination, breath sounds are equal bilaterally. There are scattered abrasions, and he is conscious.**

- **Neurologic examination is normal. He has fractures of the pelvis and ribs and an open fracture of the right femur. He has no evidence of compartment syndrome.**

Hb%	12.4 g/dL	Na	134 meq/
WBCs	11 × 10^9/L	K	6.2
Plt	434 × 10^9/L	CL	107
Ck	86,000 U/L	HCO_3	20
BUN	43 mg/dL	URINE	S.g=1.016, trace protein, 3+ hemoglobin, 2–3 RBCs/hpf, many hyaline casts/hpf
Creatinine	1.6 mg/dL		

My agenda

- Did he has AKI ?
- What are causes of AKI in trauma patients?
- What are risk factors ofAKI in trauma patients?
- How we can prevent AKI ?
- Management of crush syndrome
- Summary.

Epidemiology of AKI

⌂ 5% to 10% of hospitalized patients.

35% to 40% of patients of critically ill will develop AKI based on the RIFLE or AKIN criteria, and

5% will need renal replacement therapy.

⌂ From a Brazilian study: The prevalence of AKI in severe trauma patients was 17.3%,

Mortality, length of hospital stay and the need for ICU were significantly higher in patients who developed AKI.

⌂ AKI following surgery is an important contributor to postoperative morbidity and mortality.

André Luciano Baitellol; Gustavo Marcattoll; Roberto Kaoru Yagi,:Risk factors for injury acute renal in patients with severe trauma and its effect on mortality Risk factors for injury acute renal in patients with severetrauma and its effect on mortality. Jornal Brasileiro de Nefrologia.2013

KDIGO definition of acute kidney injury (AKI)

- **2.1.1: AKI is defined as any of the following (Not Graded):**

Increase in SCr by X 0.3 mg/dl (X 26.5 /mol/l) within 48 hours ; or

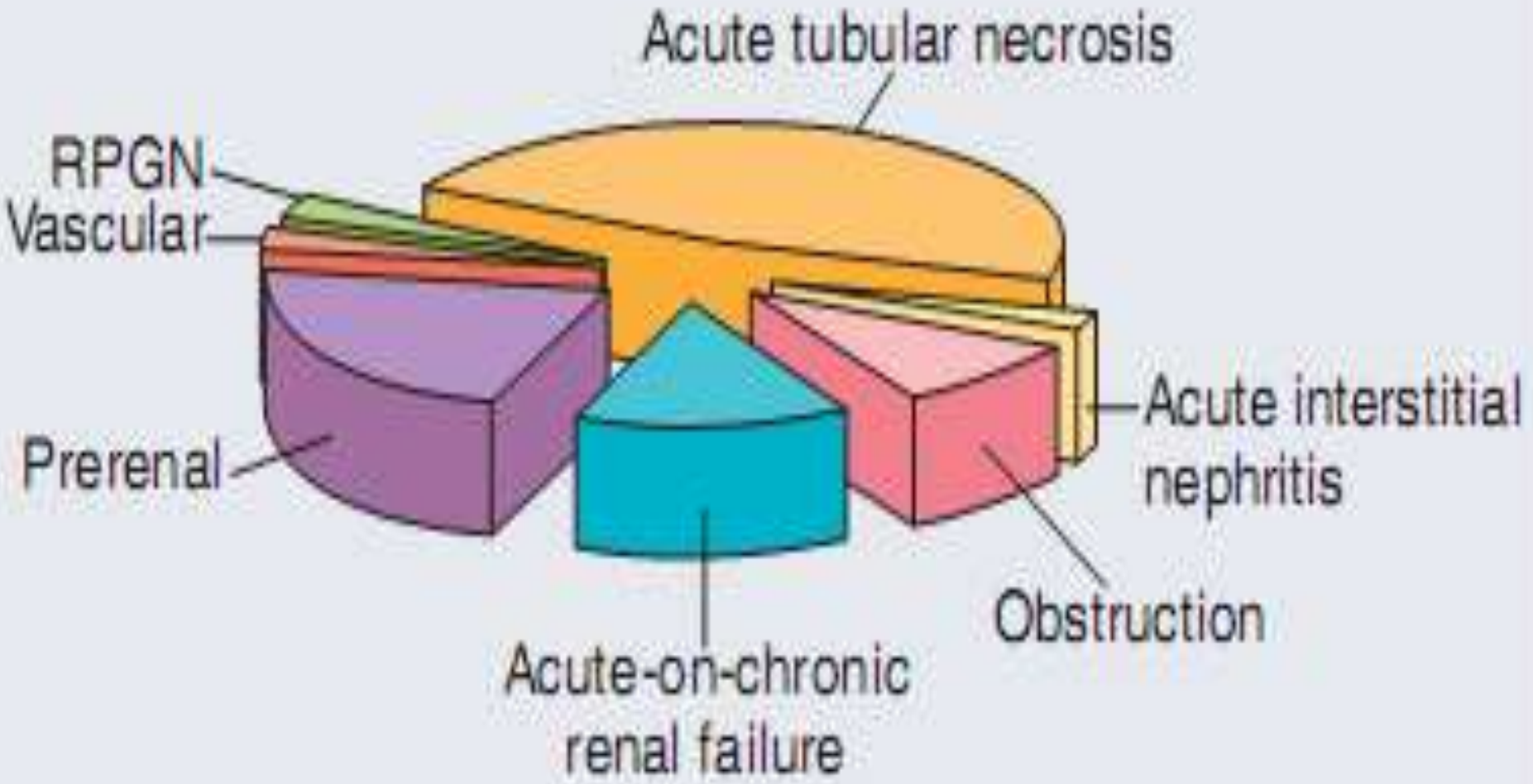
Increase in SCr to X1.5 times baseline, which is know n or presumed to have occurred within the prior 7 days; or

Urine volume 0.5 ml/kg/h for 6 hours.

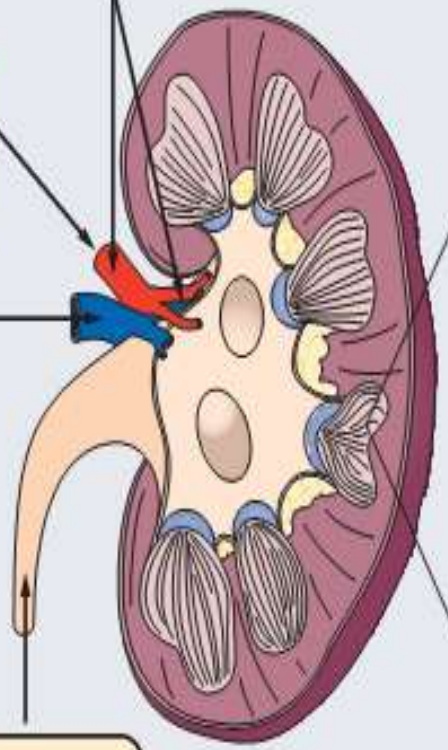
KDIGO Staging Classification of AKI

Stage	Serum creatinine (SCr) criteria	Urine output criteria
1	increase ≥ 26 µmol/L within 48hrs or increase ≥1.5 to 1.9 X reference SCr	<0.5 mL/kg/hr for > 6 consecutive hrs
2	increase ≥ 2 to 2.9 X reference SCr	<0.5 mL/kg/ hr for > 12 hrs
3	increase ≥3 X reference SCr or increase ≥354 µmol/L or commenced on renal replacement therapy (RRT) irrespective of stage	<0.3 mL/kg/ hr for > 24 hrs or anuria for 12 hrs

Causes of AKI in Hospital Setting



Causes of AKI



1. Prerenal azotemia
Hypovolemia
Cardiac failure
Hepatorenal syndrome

2. Renal artery
Renal artery occlusion
Large- or medium-vessel vasculitis

3. Small-vessel disease
Thrombotic microangiopathy
Renal atheroembolism
Small-vessel vasculitis

4. Glomerular disease
Anti-GBM disease
Lupus nephritis
Postinfectious glomerulonephritis
Infective endocarditis
Membranoproliferative glomerulonephritis
Cryoglobulinemia
IgA nephropathy/Henoch-Schönlein purpura

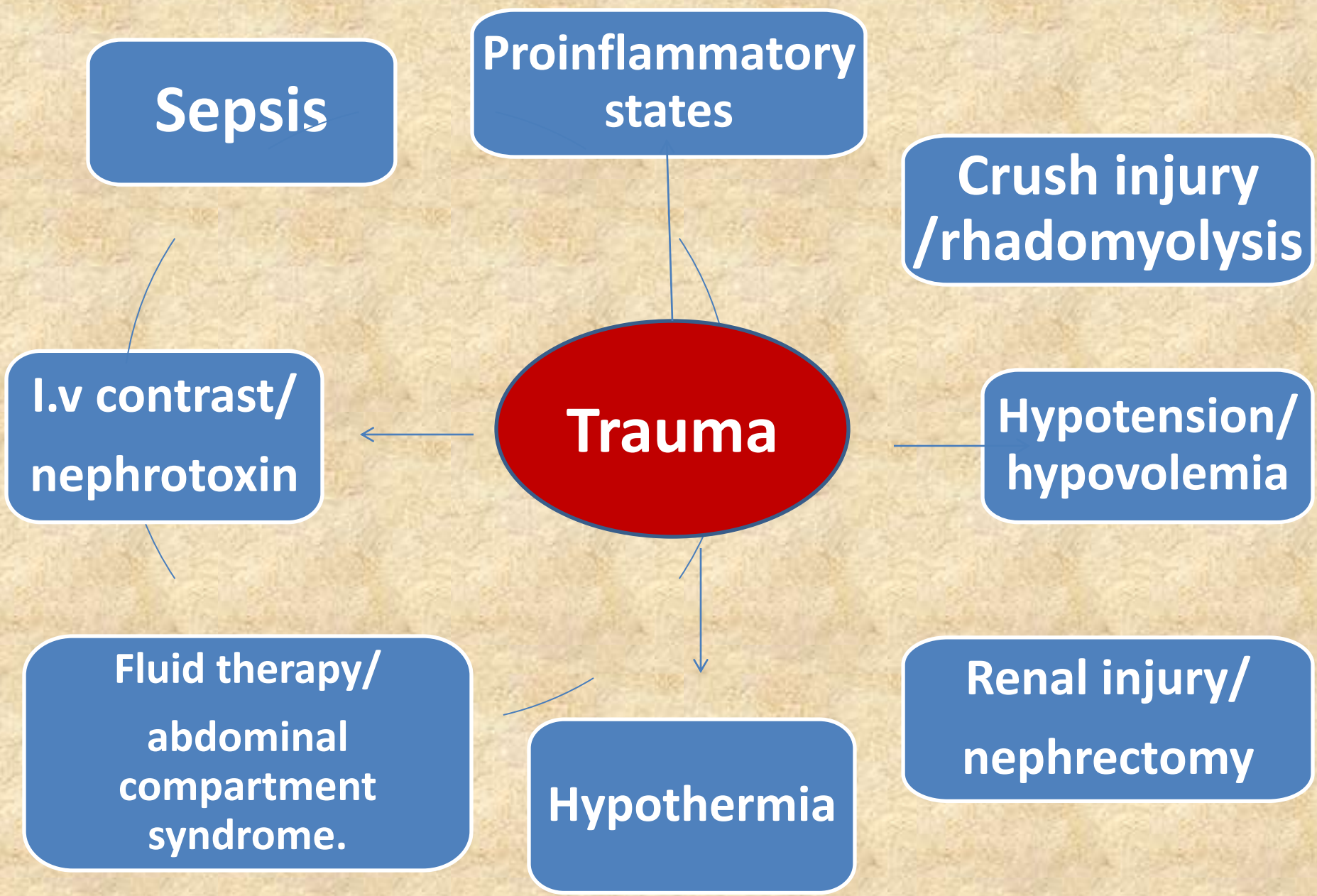
5. Acute tubular necrosis
Ischemia
Nephrotoxins
Rhabdomyolysis
Radiocontrast agents

6. Acute interstitial nephritis
Drugs
Infection
Systemic disease

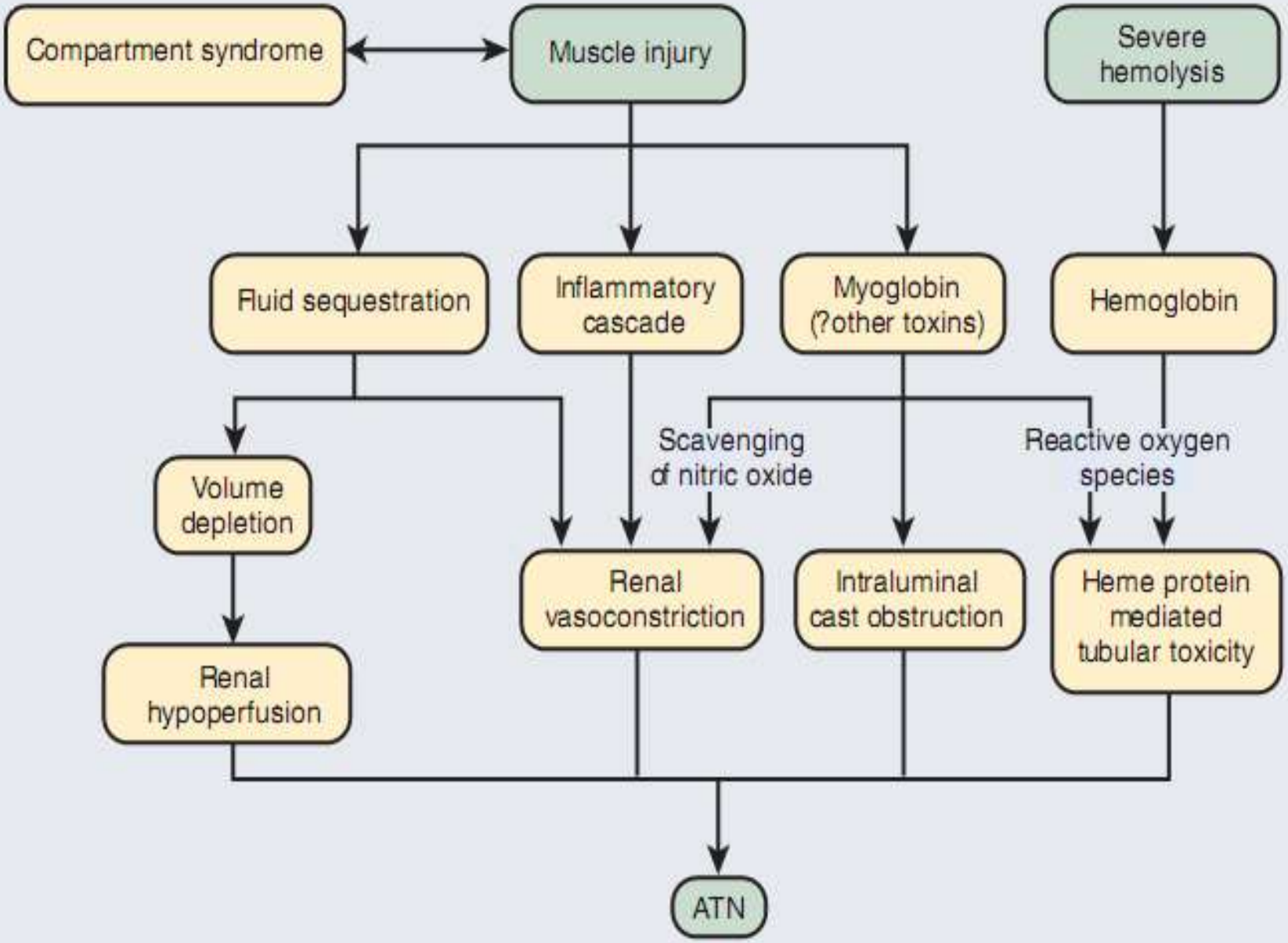
7. Intratubular obstruction
Cast nephropathy
Drugs
Crystalluria

8. Postrenal obstruction
Bladder outlet obstruction
Tumors
Renal calculi
Papillary necrosis
Retroperitoneal fibrosis

9. Renal vein
Renal vein thrombosis


Sepsis
Proinflammatory states
Crush injury /rhadomyolysis
I.v contrast/ nephrotoxin
Trauma
Hypotension/ hypovolemia
Fluid therapy/ abdominal compartment syndrome.
Hypothermia
Renal injury/ nephrectomy

Pathophysiology of Heme Pigment Nephropathy



EVALUATION OF ACASE OF AKI IN POLYTRAUMA PATIENTS

Is this acute or chronic renal failure or A/CKD?

- History and examination (DM,HTN)
- Previous creati
- Small kidneys on U/S (except diabetes)

Has obstruction been excluded?

- Complete anuria
- Palpable bladder
- Renal ultrasound

Is the patient euvolaemic?

- Pulse, JVP/CVP, post. BP, Daily BW, fluid balance disproportional increase in urea:crea ratio, urinary Na (unless on diuretics) fluid challenge

Is there evidence of renal parenchymal disease (other than ATN)?

History and examination (systemic features)
Urine dipstick and microscopy (red cells, red cell casts, eosinophils, proteinuria)

Has a major vascular occlusion occurred?

Atherosclerotic vascular disease
Renal asymmetry
Loin pain
Macroscopic haematuria
Complete anuria

Major Risk Factors for Acute Kidney Injury

Patient Factors	Medications and Agents	Procedures
Pre-existing renal dysfunction	Nonsteroidal anti-inflammatory drugs	Cardiopulmonary bypass procedures
Sepsis		Surgery involving aortic clamp
Old age (>75)	Cyclooxgenase-2 inhibitors	Increased intra-abdominal pressure
Diabetes	Cyclosporine or tacrolimus	Large arterial catheter placement with risk for atheroembolization
Hepatic failure	Angiotensin-converting enzyme inhibitors	Liver transplantation
Atherosclerosis	Angiotensin receptor blockers	Kidney transplantation
Chronic hypertension		
Perioperative cardiac dysfunction	Use of venous or arterial radiocontrast agents	
Hypercalcemia		
Renal artery stenosis		

Urinalysis

Other laboratory findings

- **Creatine kinase**
- **Potassium**
- **Calcium**
- **Phosphate**
- **Creatinine-to-urea ratio**
- **Transaminases**
- **Other**

How Do We Prevent AKI in Trauma?

Limit structural damage

–Recognize and aggressively treat pre-renal azotaemia

How?

– Maintenance of MAP in order to maintain renal perfusion pressure
    - Fluids
    - Inotropic / vasopressor support
– Limit use of ionic contrast (low osmolar)
– Limit nephrotoxic antimicrobials

Managmentof Crush-related acute kidney injury

PREVENTION

- Before and during extrication - Evidence?

- After extrication

1. - Use of bicarbonate
2. - Use of mannitol
3. - Prevention of hyperkalemia
4. - Urine output goal
5. - Total volume administered
6. - Calcium
7. - Loop diuretics

- TREATMENT OF ESTABLISHED AKI

Before and during extraction: aggressive fluid repletion(enhancerenalperfusion(minimizing ischemic injury)

- Increase UOP to wash out the obstructing casts.
- Preventive therapy appears to be less effective after the first 6 to 12 hours(AKI established)

- 7 patients with crash syndrome received alkaline diuresis immediately non of them developed AKI(Ron, et al1984).
- One patient who did not receive prophylactic volume repletion developed AKI and required hemodialysis [Reis, and Michaelson1986].

Type of fluid

- Isotonic saline rather than isotonic bicarbonate be administered because.?
- Isotonic saline should initially be given at a rate of 1 L/hour (10 to 15 mL/kg of body weight per hour) while the victim is still under the rubble. After 2 liters - the rate of administration should be decreased to 500 mL/hour to avoid volume overload.
- Ringer solution :avoided ?

Amount of fluid

- Age
- Body mass index
- Trauma.
- Amount of presumed fluid loss.

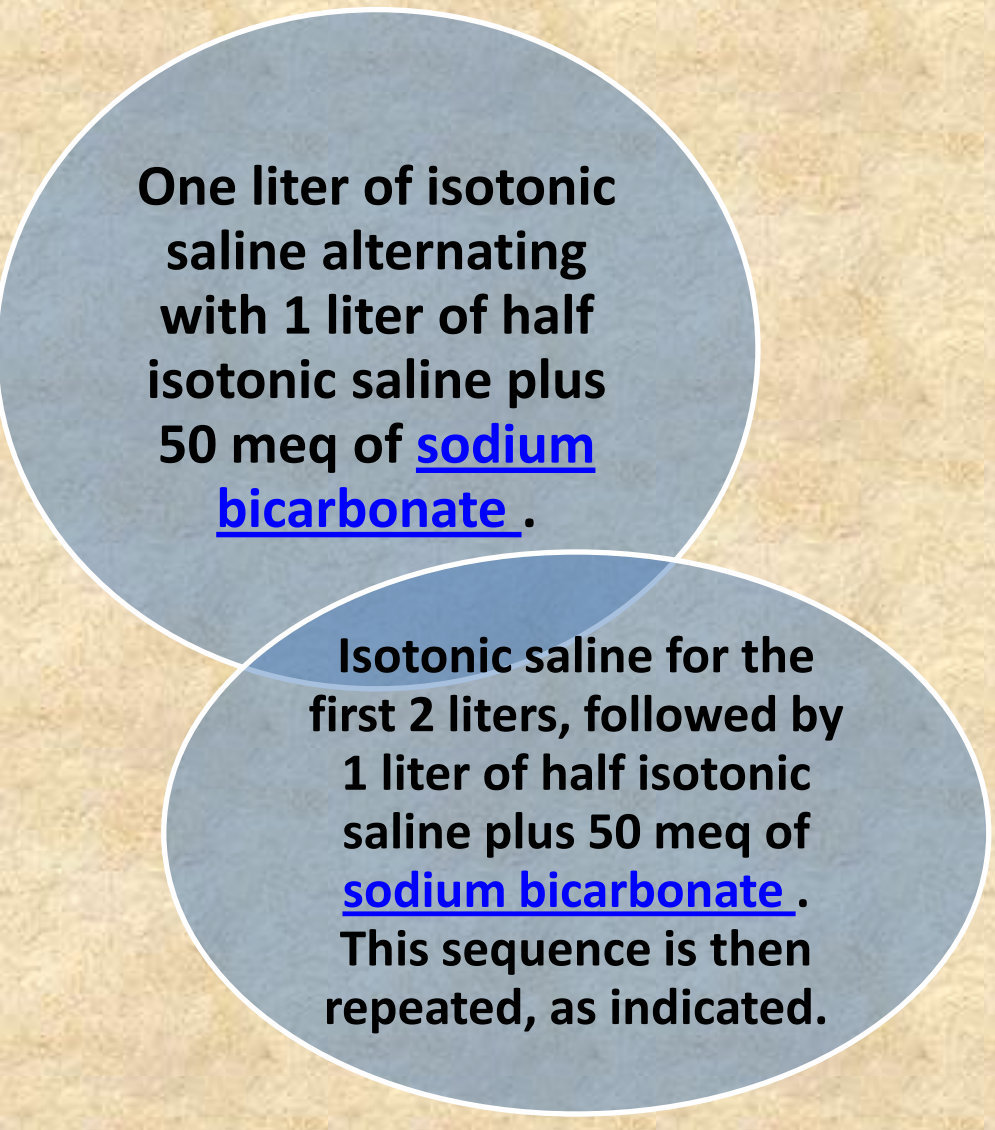
After extrication

Use of bicarbonate

- Renal Disaster Relief Task Force (RDRTF) in an attempt to achieve a forced alkaline diuresis.
- The rationale : urine pH > 6.5 may prevent heme-protein precipitation with Tamm-Horsfall protein, intratubular pigment cast formation, uric acid precipitation, correct metabolic acidosis and reduce hyperkalemia

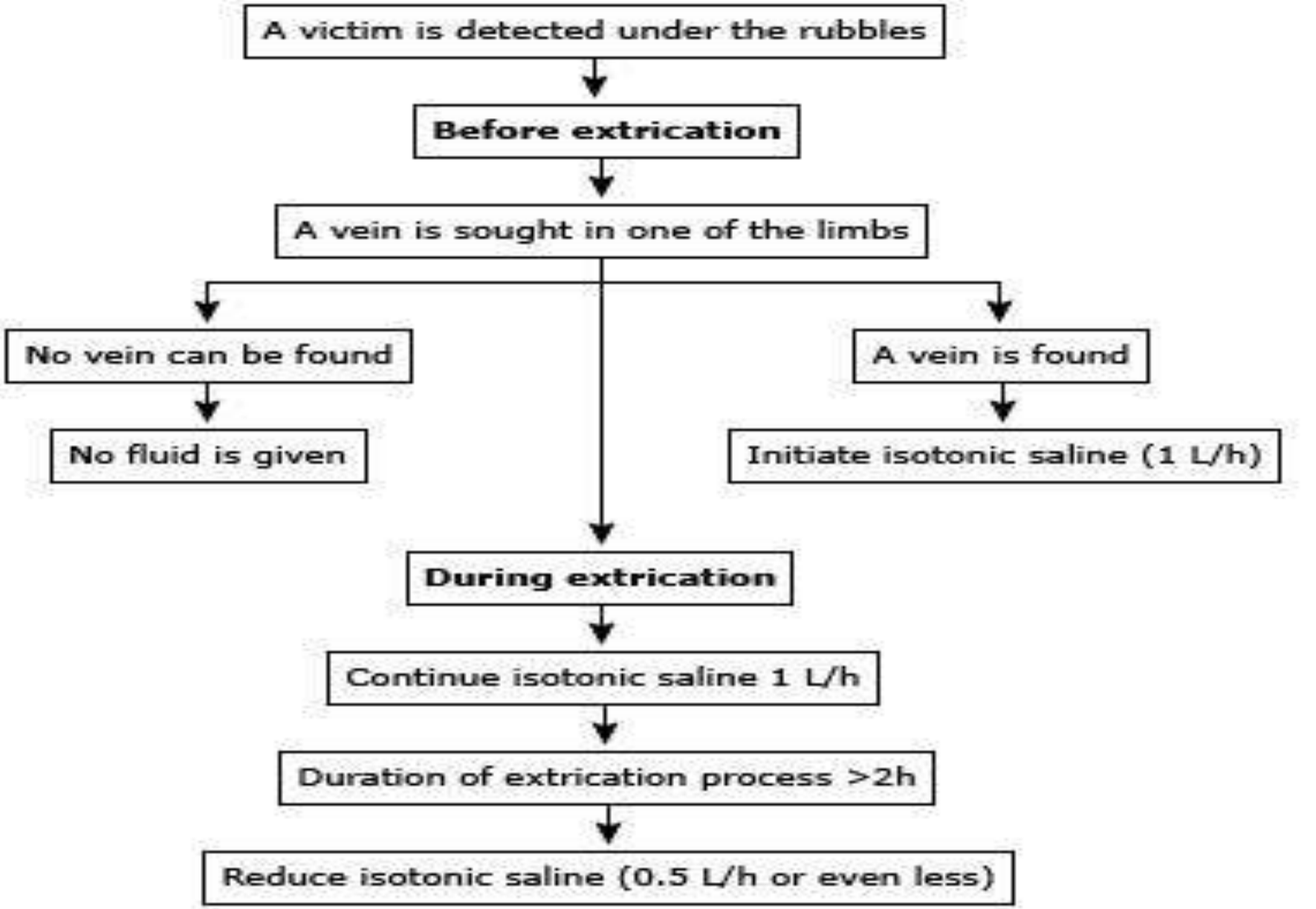
- Despite these potential benefits, there is no clear clinical evidence that an alkaline diuresis is more effective than a saline diuresis in preventing acute kidney injury, as no direct comparative trial has been performed.

The optimal regimen and rate of administration of bicarbonate are unknown

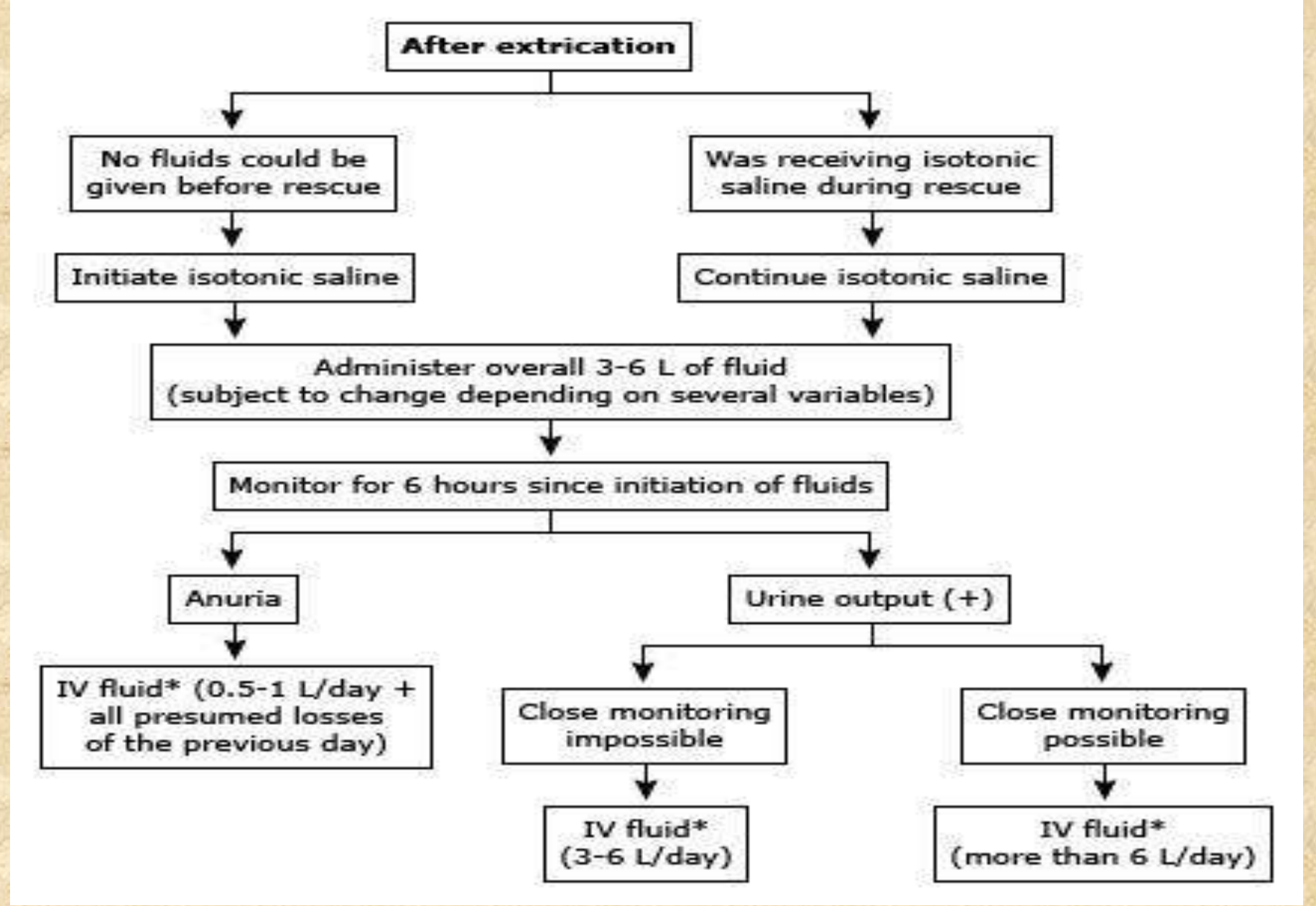


Which is best?

- Depends in part upon the general clinical and biochemical condition of the patient and the blood pH.
- The rate of fluid administration with either regimen is based upon the ability to attain urinary output goals, and assessment of volume status. In general, we administer the intravenous solution at 500 mL/hour for the first 24 hours as long as there is no evidence of fluid overload and the patient can be closely monitored.



Fluid administration protocol in adults before and during extrication for entrapped victims of mass disasters.



Algorithm for fluid resuscitation to prevent crush-related AKI in entrapped victims of mass disasters early after extrication.

Nephrol Dial Transplant 2012

Prevention of Hyperkalemia

Majority of patients develop hyperkalemia due to rhabdomyolysis.may seen before AKI

Hyperkalemia may be a lifethreatining condition.

Avoid k-containing solution.

Sodium Polystyrene Sulfonate – Calcium Polystyrene Sulfonate

- If serum potassium concentration cannot be measured due to field conditions, ECG

Use of Mannitol

Mechanism of action to protect againt heme-pigment induced ATN: unclear?

Mannitol should only be used if close monitoring is possible. Mannitol is contraindicated in patients with oligoanuria.

Adequate UOP(defined as >20 mL/hour), 50 mL of 20 % mannitol (1 to 2 g/kg per day [total, 120 g], given at a rate of 5 g per hour) +to each liter of fluid providing an increase in urine output is demonstrated following a test dose of mannitol.

The available retrospective series, most of which are uncontrolled, report conflicting results regarding the effectiveness of mannitol plus bicarbonate in preventing heme pigment-induced AKI

Urine Output Goal

- Insertion of Foley catheter.
- IVF -maintain UOP 200-300mL/h
- TILL disappearance of myoglobinuria
- Frequent assess to avoid overload .
- Central venous pressure (CVP)
- Close monitoring of fluid intake and output, and body weight

Total Volume Administered

- Depends upon the clinical scenario. up to 12 L/day to an adult weighing 75 kg and with appropriate urine response.
- More cautious volume repletion is also warranted in victims who are prone to cardiac failure, such as the elderly, and in those who are anuric
- Analysis of the Bingol earthquake demonstrated that dialysis was avoided in many patients with crush syndrome by administering more than 20 L of fluid per day to each patient (Gunal, et al., 2004)

Calcium

- Calcium :only for symptomatic hypocalcemia or severe hyperkalemia, because early deposition of Ca muscle is followed by hypercalcemia later in the injury process

Loop diuretics

- No impact on outcome in AKI In rhabdomyolysis, loop diuretics may worsen the hypocalcemia, since they induce calciuria
- and may increase the risk of cast formation.
- Judicious use of loop diuretics may be justified in elderly patients, especially if volume overloaded.

TREATMENT OF ESTABLISHED AKI

- No specific therapy once the patient has developed AKI.

Dialysis (as usual indications),
volume **o**verload
hyper**ka**lemia,
severe a**cid**emia,
and **Uremia**.

- **NB: HD may be indicated in patients with crush syndrome, given the high risk of fatal hyperkalemia**

Dialysis Modalities

I.HD : is suggested over other renal replacement modalities

Compared with other modalities, I.HD is most efficient at removing potassium

Peritoneal dialysis (PD):limited due to technical and logestic problem

CRRT: hemodynamically unstability mainly

Summary of management of acute crushsyndrome

1. Hypovolemia + myoglobin secondary to rhabdomyolysis →acute tubular necrosis (ATN), →(AKI). AKI →major source of morbidity and mortality in natural or man-made disasters
2. Starting intravenous fluid replacement prior to and during extrication of the victim whenever possible.
3. Isotonic saline rather than an isotonic alkaline solution.

4) We suggest administering fluid at 1 L/hour initially. After 2 liters are given, the rate of administration should be decreased to 500 mL/hour to avoid volume overload.
5) Severe hyperkalemia is relatively common.
6) As Ringer's lactate, are contraindicated.

Summary of management of Rhabdomyolysis

- After extraction isotonic saline alternating to an isotonic bicarbonate solution.
- monitoring of S HCO_3,Ca, K, and serum and urine pH.
- If UOP>20 mL/hour, adding mannitol to the intravenous alkaline solution providing an increase in urine output is demonstrated following a test dose

- Discontinuing mannitol if the desired diuresis cannot be achieved (approximately 200 to 300 mL/hour.
- If the UOP goal is achieved, continue fluid therapy until the disappearance of myoglobinuria.
- Patients with symptomatic hypocalcemia or severe hyperkalemia may require calcium supplementation
- Dialysis is initiated for the usual indications

Nephrotoxins

- NSAIDs
- Aminoglycosides
- Amphotericin
- Penicillins
- Acyclovir
- Cytotoxics
- Radiocontrast dye

Dennen P, Douglas I, Anderson R,: Acute Kidney Injury in the Intensive Care Unit: An update and primer for the Intensivist. *Critical Care Medicine* 2010; 38:261-275.

Conclusions

- AKI is common following major trauma.
- AKI has an impact on mortality

- Can Aki be prevented in trauma,,, ?,
- just avoid trauma
- IF NO ?

  Maintain renal perfusion,

  Limitation use of nephrotoxic contrast dye

  Limite the use of nephrtotoxic drugs.

  AKI following rhabdomyolysis is rare

Thank you for your attention

Indications – Hyperkalemia

- Hyperkalemia can be rapidly fatal
  - No specific threshold, although $[K^+] < 6.5$mmol/L are unlikely to cause cardiac toxicity
  - Between 50 and 200 mmol of K^+ can be removed over a 4 hour iHD treatment
  - Between 10-20 mmol of K^+ can be removed every hour during CRRT
  - Initiation of ARRT based on the absolute level and rate of increase of $[K^+]$ and the patient's overall condition